



Anthropometric indicators and body composition as determinants of healthy ageing in older adults in a rural area of Ecuador

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ABSTRACT

Premature biological aging manifests as physiological deterioration exceeding chronological age, substantially increasing morbidity risk, particularly in rural settings where body composition is a critical determinant of functional health. This research aimed to analyze the association between anthropometric indicators, body composition, and healthy aging in older adults from a rural Ecuadorian parish. A quantitative, correlational, cross-sectional study was conducted with 313 participants. Weight, height, Body Mass Index (BMI), and body compartments (adipose and muscle tissue) were evaluated using medical-grade electrical bioimpedance. Findings revealed a 2.91-year gap between biological age (77.21 years) and chronological age (74.30 years), evidencing accelerated aging. A pattern of sarcopenic obesity predominated (mean BMI: 27.64; body fat: 39.67%; muscle mass: 20.50 kg). Dispersion analysis demonstrated that BMI increases correspond mainly to adipose tissue ($R^2=0.211$) rather than muscle reserve ($R^2=0.033$), indicating critical muscle frailty. It is concluded that BMI alone is insufficient to predict functional health, and the imbalance between adiposity and musculature compromises the physical autonomy of this population.

Keywords: *Healthy Aging; Body Composition; Anthropometry; Premature Aging; Rural Population; Sarcopenic Obesity.*

INTRODUCTION

The dysregulation of homeostasis and the progressive degeneration of physiological functions are central manifestations of ageing, understood as a biological process of a multifactorial and progressive nature (Mesnage, 2025; Guo et al., 2022). From a molecular perspective, this phenomenon is due to a set of interconnected mechanisms or "hallmarks", including mitochondrial deterioration, telomere shortening, cellular senescence, and genomic instability (Maldonado et al., 2023; Proal & VanElzakker, 2025; Li et al., 2024). These factors not only determine the variability in the speed of systemic deterioration but also interact dynamically with the immune system, promoting a state of chronic low-grade inflammation and modifying hematopoiesis, which substantially increases susceptibility to age-associated diseases (McHugh et al., 2025; Bruserud et al., 2022). From the pathophysiological point of view, the changes present a wide

heterogeneity, which explains the enormous variability observed in health and functionality conditions during old age.

Contemporary scientific literature establishes a precise distinction between chronological and biological age; the latter being a more accurate indicator of the functional status of the person (Forrester et al., 2024), since it covers various domains of aging, such as adaptation to stressors of a metabolic, psychosocial and physical nature. In this sense, accelerated biological aging has emerged as a crucial risk element for the development of pathologies such as cardiovascular diseases, type 2 diabetes mellitus, and cardiometabolic multimorbidity (Zhang et al., 2025; Jiang et al., 2024). It is essential to consider that the excessive and progressive accumulation of cellular and metabolic damage converges with a low level of physiological reserve, increasing the probability of dependence and functional deterioration (Jiang et al., 2024). Recent data suggest that this biological acceleration also affects mental health, increasing the risk of depression and anxiety in older people (Gao et al., 2023). In this context, cellular senescence could be exacerbated by the prolonged release of stress hormones and alterations in the hypothalamic-pituitary-adrenal axis, accelerating tissue damage (Qin et al., 2024) and enhancing a vicious circle between chronic stress, inflammation and accelerated aging.

The rate of ageing is not determined solely by genetic factors, but is strongly influenced by the "exposome", which comprises the totality of environmental exposures, from air pollution to the work environment (Pandics et al., 2023; Di Ciaula & Portincasa, 2020). Chronic accumulated exposures throughout life can alter the biological mechanisms of aging and condition health status in advanced stages. In addition to environmental factors, social determinants, such as living in poverty, have demonstrated the ability to induce biological acceleration measurable by both epigenetic clocks and clinical biomarkers (Dalecka et al., 2024; Ibáñez de Opakua et al., 2025). Social inequality, limited access to health services, and food insecurity act as catalysts for vulnerability in old age. In this scenario, adipose tissue acts as an endocrine organ that, as it ages, undergoes a dysfunctional redistribution that promotes insulin resistance and frailty, complicating clinical conditions such as geriatric delirium (Ou et al., 2022; Bellelli et al., 2024). These bodily alterations directly influence the functional capacity and autonomy of older adults.

To mitigate these effects, interventions have been proposed ranging from the correction of systemic mechanisms through complex physiotherapy to lifestyle changes that optimize body composition (Korchazhkina et al., 2024). The evaluation of simple anthropometric indicators allows an approximation of nutritional and functional status, especially in contexts of limited resources. However, in rural populations of Ecuador, the association between anthropometric indicators and healthy aging has not yet been fully characterized under these new biological paradigms. The limited local evidence makes it difficult to design preventive strategies adapted to the rural sociocultural context. Therefore, the objective of this study is to analyze how body composition and anthropometric indicators are associated with healthy aging in older adults in a rural Ecuadorian parish.

METHODOLOGY

This research is defined as a quantitative study, with a descriptive and correlational scope, cross-sectional, designed to analyze the relationship between nutritional status and the senescence process in the rural context. This design made it possible to describe the anthropometric and body composition characteristics of the elderly population at a specific time, as well as to explore the associations between these variables without establishing direct causal relationships.

The population consisted of 313 older adults residing in a rural parish in Ecuador, selected through a non-probabilistic convenience sampling, considering the accessibility, availability, and availability of the participants during the data collection period. This type of sampling was appropriate given the logistical limitations of rural environments and the absence of an updated census record of the elderly population.

The inclusion criteria included people of both sexes, with a minimum age of 65 years, who permanently resided in the parish and who agreed to participate voluntarily through informed consent. Individuals with severe physical disabilities that prevented standing or taking anthropometric measurements were excluded, as well as those with diagnosed cognitive impairment that limited the understanding of instructions and the correct execution of measurements.

A structured anthropometric record sheet, previously designed to record sociodemographic and physical variables, and a medical-grade electrical bioimpedance body composition analyzer were used for data collection. The application of this instrument allowed to obtain precise values of weight, height, body mass index (BMI), body fat percentage, skeletal muscle mass in kilograms and the estimation of body age. The measurements were carried out following standardized protocols, guaranteeing adequate conditions such as relative fasting, removal of metal objects and correct posture, in order to minimize possible measurement biases.

The statistical analysis was carried out using the IBM SPSS Statistics software, initially applying descriptive statistics with the calculation of absolute and relative frequencies for categorical variables, as well as means, medians, standard deviations and ranges for quantitative variables, with the aim of comprehensively characterizing the sample and detecting missing or outliers. Prior to the correlational analyses, the assumptions of normality and homogeneity of the variables were verified by means of exploratory and graphical procedures. Subsequently, the strength of association between the variables was evaluated using Pearson's correlation coefficient and the coefficient of determination (R^2) for the dispersion analyses, allowing quantifying the link between BMI and the fat and muscle components, as well as the agreement between chronological age and estimated biological age. These analyses facilitated the identification of patterns of accelerated biological aging and their relationship with alterations in body composition. The results were organized in descriptive tables, histograms and scatter plots, guaranteeing rigor in the interpretation and respecting the ethical principles of confidentiality and anonymity.

RESULTS

General characteristics of the population

The analysis of the data reveals that the population of older adults evaluated presents a premature biological aging, with an average body age of 77.21 years, which significantly exceeds its chronological age of 74.30 years. This physiological deterioration is based on an unfavorable body composition, characterized by a BMI of 27.64 and a high fat percentage of 39.67%, which, added to a reduced muscle mass of just 20.50 kg, describes a profile of sarcopenic obesity of high metabolic risk. The variability of the sample, with cases of morbid obesity and critical levels of muscle malnutrition, shows a functional vulnerability that compromises the autonomy of this group, making it imperative to implement comprehensive interventions to mitigate the impact of this imbalance on their health and quality of life (Table 1).

The research shows premature biological aging and a sarcopenic obesity profile in older adults, characterized by excess body fat compared to deficient muscle mass. This metabolic and functional imbalance, aggravated by critical frailty values, seriously compromises the physical autonomy of the sample. Therefore, it is urgent to implement comprehensive nutrition and adapted exercise interventions to reverse the observed physiological deterioration.

Table 1. Descriptive statistics of the anthropometric variables of the population studied

Statistic	Chronological age	Body Age	BMI (kg/m ²)	% Body Fat	Skeletal Muscle Mass (kg)
N Valid	313	276	302	302	295
Media	74,30	77,21	27,64	39,67	20,50
Medium	74,00	77,00	27,08	40,20	19,80
Desv. East.	7,58	8,63	4,68	8,85	5,43

Minimum	65	56	18,73	19,20	2,6
Maximum	95	99	44,26	70,00	37,6

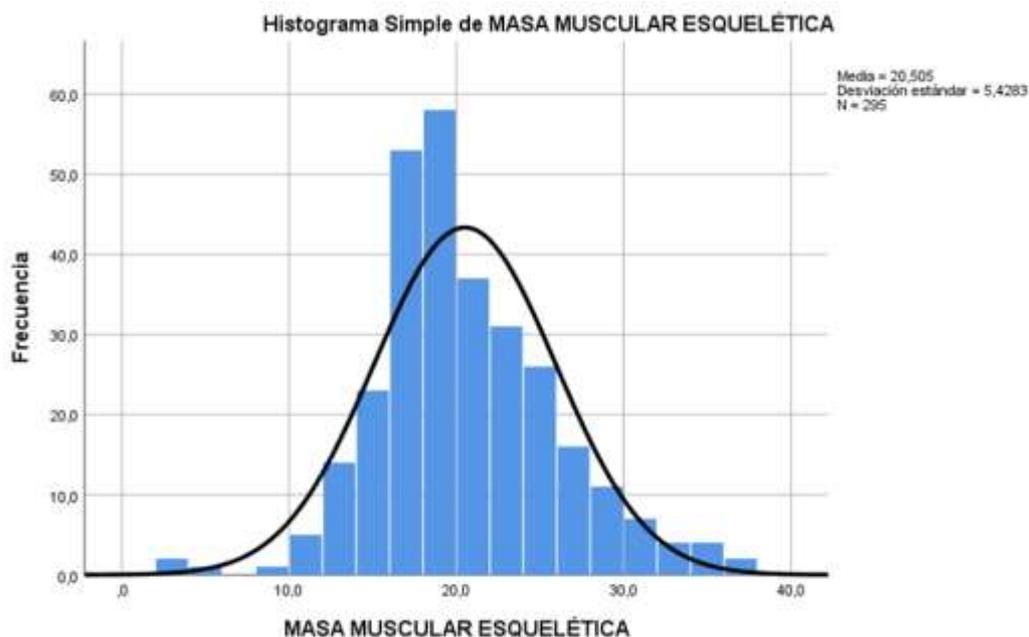
Source: Authors.

Distribution of skeletal muscle mass

The research concludes that the population evaluated presents premature biological aging (77.21 years compared to 74.30 chronological years), closely linked to a sarcopenic obesity profile. This state is defined by the coexistence of excess body fat (39.67%) and deficient muscle mass (20.50 kg), where weight gain is associated with adipose tissue ($R^2=0.211$) and not with protective muscles ($R^2=0.033$). The negative correlation between muscle and fat (-0.355), together with critical levels of frailty, shows a high functional vulnerability that makes it urgent to implement body recomposition programs to preserve the autonomy of the elderly.

The research shows accelerated aging and sarcopenic obesity, defined by excess fat and muscle deficiency that compromise physical autonomy. The data confirm that body weight is associated with adipose tissue and not with protective muscles, generating a high functional vulnerability. Therefore, it is urgent to implement body recomposition programs that prioritize muscle strengthening to mitigate metabolic risks in the elderly.

FIGURE 1 Distribution of skeletal muscle mass. In the original Spanish language Determination of Accelerated Biological Aging and Risk of Sarcopenic Obesity

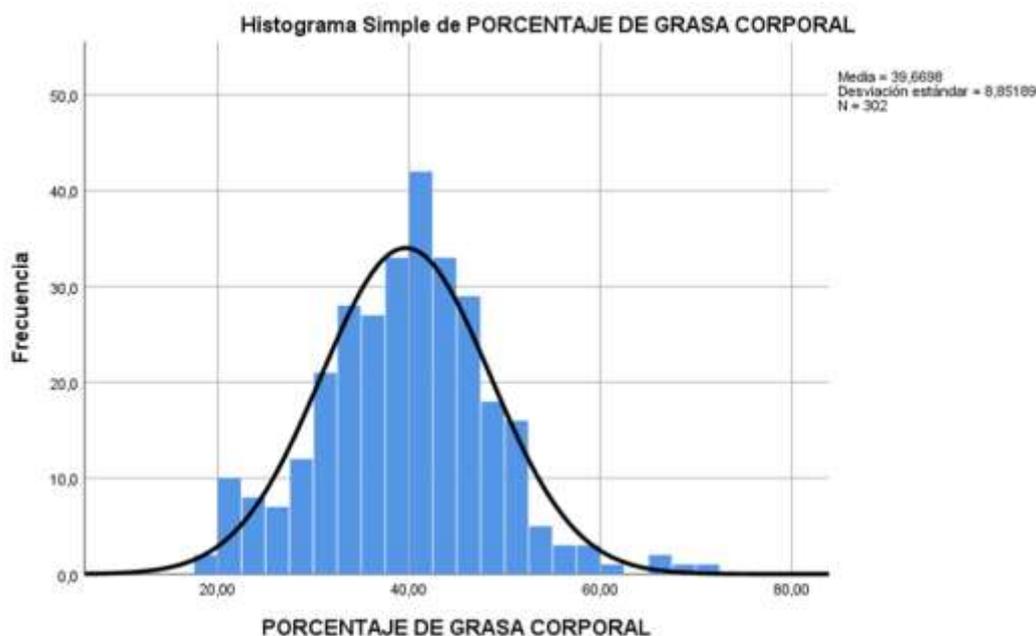


The study concludes that the population has accelerated biological aging (77.21 body years) and a sarcopenic obesity profile, defined by excess fat (39.67%) and deficient muscle mass (20.50 kg). The data reveal that physiological deterioration and critical levels of muscle frailty seriously compromise the physical autonomy and metabolic health of older adults. This vulnerability is compounded by a body composition where weight is predominantly associated with adipose tissue rather than protective

musculature. Therefore, it is imperative to implement body recomposition programs that prioritize muscle strengthening to mitigate these risks. These interventions are essential to preserve functionality and improve the overall quality of life in this age group.

The study shows accelerated aging and sarcopenic obesity. This condition links weight to adipose tissue and not to the protective musculature, seriously compromising autonomy and metabolic health. Therefore, it is imperative to implement muscle-strengthening and body recomposition programs to mitigate the functional vulnerability of the elderly.

FIGURE 2 Distribution of body fat percentage. In the original Spanish language



Relationship between BMI and body composition variables

The study shows accelerated biological aging driven by a sarcopenic obesity profile, where excess fat and muscle deficiency increase metabolic risk. The data confirm that weight gain is linked to adipose tissue ($R^2 = 0.211$) and not to protective muscles, generating a high functional vulnerability. The presence of critical levels of muscle mass of just 2.6 kg seriously compromises the physical autonomy of older adults. Therefore, it is imperative to implement body recomposition programs that prioritize muscle strengthening to mitigate risks and improve quality of life. These interventions are essential to preserve integral functionality in this age group.

The study reveals accelerated aging and sarcopenic obesity that compromise physical autonomy. The low muscle correlation against excess fat confirms a high functional and metabolic vulnerability. Therefore, it is urgent to implement muscle-strengthening and body recomposition programs to improve the quality of life of the elderly.

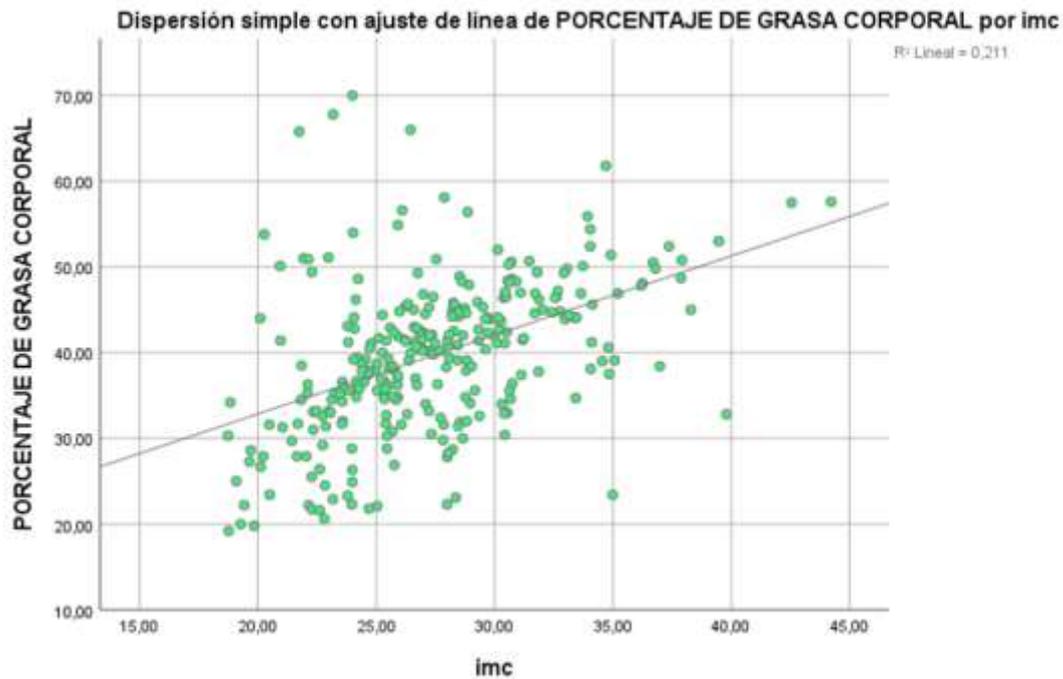


Figure 3: Relationship between body mass index and body fat percentage. In the original Spanish language

Accelerated Biological Aging and Risk of Sarcopenic Obesity: Impact on Autonomy

The study concludes that the population presents accelerated biological aging and a sarcopenic obesity profile. The data reveal that body weight is linked to adipose tissue ($R^2 = 0.211$) and not to protective muscles ($R^2 = 0.033$), generating critical levels of muscle fragility of up to 2.6 kg. This condition seriously compromises the physical autonomy and metabolic health of older adults. Therefore, it is imperative to implement body recomposition programs that prioritize muscle strengthening to mitigate functional risks. These interventions are essential to preserve functionality and improve the overall quality of life in this age group.

The study shows accelerated aging and sarcopenic obesity that compromises the autonomy of the elderly. Since weight is linked to adipose tissue and not to muscle, it is urgent to implement muscle-strengthening and body recomposition programs. These interventions are key to mitigating functional risks and improving quality of life.

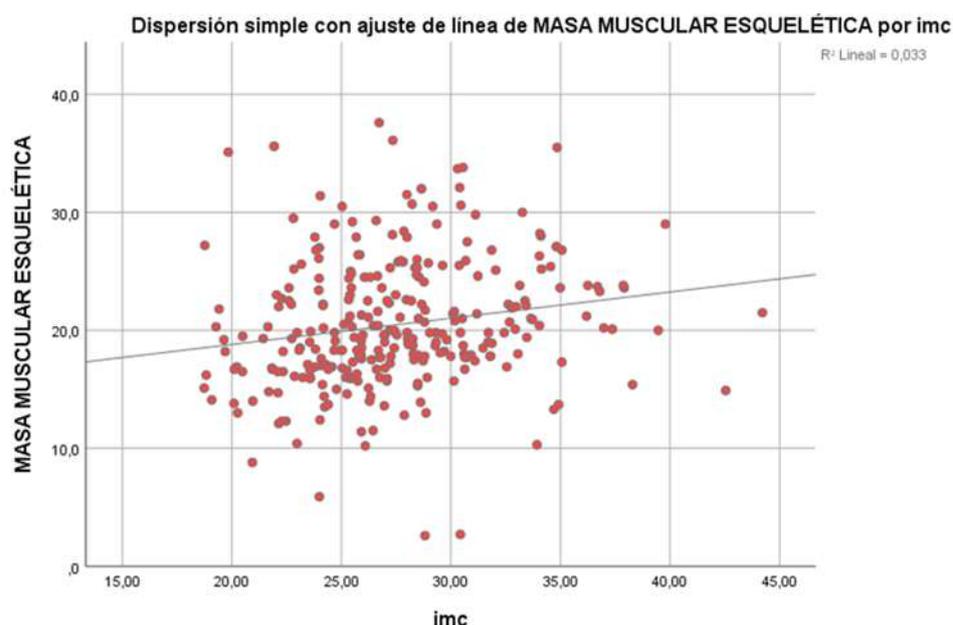


Figure 4: Relationship between body mass index and skeletal muscle mass. In the original Spanish language

Analysis of Physiological Impairment and Risk of Sarcopenic Obesity

The study reveals accelerated aging driven by a sarcopenic obesity profile, where excess fat and muscle deficiency elevate metabolic risk. The data confirm that the increase in weight is linked to adipose tissue ($R^2 = 0.211$) and not to protective muscles ($R^2 = 0.033$), generating a high functional vulnerability. Therefore, it is urgent to implement body recomposition programs focused on muscle strengthening to preserve autonomy and improve the quality of life of the elderly.

The research shows accelerated aging driven by a sarcopenic obesity profile, where excess fat and deficient muscle mass compromise physical autonomy. The data confirm that weight gain is linked to adipose tissue and not to protective muscles, generating a high functional vulnerability in the elderly. It is imperative to implement body recomposition interventions that prioritize muscle strengthening to mitigate these metabolic risks and improve quality of life.

Table 2. Distribution of the Median and Interquartile Range of Fat Percentage according to the Aging Classification

Classification of Aging	Median (%)	Interquartile Range (IQR)
Healthy	34,45	37,55 -- 44,15
Expected	40,50	34,02 -- 45,75
Accelerated	39,15	34,50 -- 44,52

Source: Authors.

Determination of Biological Aging and Risk Profile

The study concludes that the population presents an accelerated biological aging and a sarcopenic obesity profile, where excess fat does not translate into a protective muscle reserve ($R^2 = 0.033$). This

disproportion, evidenced by deficient muscle mass and critical levels of frailty, seriously compromises the physical autonomy of older adults. Therefore, it is urgent to implement body recomposition programs focused on muscle strengthening to reduce functional vulnerability and improve the overall health of the group.

The research concludes that the population has accelerated biological aging and sarcopenic obesity, characterized by excess fat and deficient muscle mass. The data confirm that weight gain is associated with adipose tissue and not with protective muscles, generating a high functional vulnerability. Therefore, it is urgent to implement body recomposition programs that prioritize muscle strengthening to mitigate metabolic risks and preserve the autonomy of the elderly.

Table 3. Pearson Correlation Matrix between Chronological Age, Body Age, and Body Composition Indicators

Variable	Age	Body Age	IMC	% Body Fat	Muscle Mass
Age	1,000	0,835	-0,044	0,189	-0,329
Body Age	0,835	1,000	0,058	0,339	-0,349
IMC	-0,044	0,058	1,000	0,478	0,238
% Body Fat	0,189	0,339	0,478	1,000	-0,355
Muscle Mass	-0,329	-0,349	0,238	-0,355	1,000

Source: Authors.

DISCUSSION

The results of this study reveal a critical phenomenon of accelerated biological aging in the elderly of the rural parish evaluated. The discrepancy found between chronological age ($\bar{x} = 74.30$ years) and body age ($\bar{x} = 77.21$ years) suggests a premature physiological deterioration that exceeds the natural process of senescence. This gap of almost three years of additional ageing should not be considered an isolated event, but a direct consequence of an unfavourable body composition, characterised by an overweight BMI (27.64), a high percentage of fat (39.67%) and a worrying deficiency of skeletal muscle mass (20.50 kg). This phenotypic imbalance indicates that excess adipose tissue acts as a potent inducer of cellular senescence, promoting a pro-inflammatory systemic environment that accelerates the wear and tear of vital systems and prematurely depletes the individual's homeostatic reserves.

The finding of a body age higher than the chronological age coincides with what was proposed by Ventura et al. (2025), who argue that the acceleration of phenotypic age is a robust indicator of frailty in older adults. In this context, the strong correlation found in our study between chronological and body age ($r = 0.835$) reinforces the idea that biological factors and lifestyle determinants are negatively driving the health profile of Ecuadorian rural older adults. Toyoshima et al. (2022) have shown that the difference between "physical fitness age" and chronological age is closely associated with obesity and hyperglycemia, which explains why our population, with a high BMI, presents accelerated aging. In rural areas, visceral fat accumulation increases insulin resistance, which not only alters glucose metabolism but also interferes with muscle protein synthesis. In addition, this metabolic state favors the sustained release of reactive oxygen species, exceeding the endogenous antioxidant capacity and damaging the integrity of cell membranes in the musculoskeletal system.

The coexistence of high adiposity and low muscle mass in the sample configures a profile of sarcopenic obesity. This state not only represents an aesthetic change, but a complex pathological process of fatty infiltration into the contractile tissue. According to Wang et al. (2025), sarcopenic obesity significantly impacts biological aging because adipose tissue exacerbates inflammatory processes that degrade muscle. This aligns with our results, where dispersion analysis showed that weight gain ($R^2 = 0.211$ with body fat)

does not translate into increased muscle reserve ($R^2 = 0.033$), evidencing a disconnect between caloric intake and tissue functionality.

The loss of muscle mass (sarcopenia) is the central axis of this deterioration. Gustafsson and Ulfhake (2021) point out that the origin of this age-induced disorder is multifactorial, involving everything from neurohumoral changes to the denervation of muscle fibers. Molecular mechanisms, such as damage to mitochondrial DNA, accelerate this loss of skeletal muscle tissue (Luo et al., 2024), while alterations in signaling pathways (such as Akt1/2 deletion) not only cause osteosarcopenia but also dramatically reduce life expectancy (Sasako et al., 2022). Early identification of these pathways is crucial in rural areas, where physical dependence often precipitates social isolation. The degradation of the motor unit in these patients implies a loss of precision in movement, exponentially increasing the incidence of falls and fractures that seriously compromise survival.

The functional vulnerability of the participants, with critical cases of only 2.6 kg of muscle mass, increases the risk of chronic diseases. Damluji et al. (2023) stress that sarcopenia is an independent risk factor for cardiovascular disease, while Knoedler et al. (2023) have demonstrated through meta-analyses that the presence of sarcopenia dramatically worsens clinical outcomes in surgical patients. The gut microbiota also seems to play a mediating role; recent studies suggest that the dietary index for the microbiota is associated with muscle health, linking nutrition directly to biological age (Zhang et al., 2025). In rural settings, this relationship is vital, because as Swan et al. (2022) indicate, socioeconomic disadvantages are usually associated with a higher probability of sarcopenia in older adults living in the community. It is highly likely that prolonged exposure to poorly diversified diets, added to the environmental stress of the field, has altered the intestinal symbiosis of these adults, feeding a cycle of “inflammaging” or chronic inflammation that rapidly consumes protein reserves.

To mitigate this accelerated aging, the literature suggests innovative therapeutic approaches. Selvais et al. (2024) explore the use of molecules such as AdipoRon to extend muscle health, while Wang and Zhou (2025) emphasize the importance of interventions that address both sarcopenia and cachexia through specific molecular mechanisms. Likewise, the optimization of energy metabolism (He et al., 2023) and the approach to epigenetic mechanisms (Antoun et al., 2022) emerge as critical ways to slow down musculoskeletal deterioration (Cai et al., 2024). These strategies must be adapted to the rural context, prioritizing mechanical loading exercises that stimulate neuroplasticity and muscle regeneration through local resources.

CONCLUSIONS

The research allows us to conclude that the population of older adults in the rural parish evaluated experiences accelerated biological aging, where the average body age of 77.21 years significantly exceeds the chronological age of 74.30 years, which shows a premature physiological deterioration and a greater susceptibility to chronic pathologies. This systemic wasting is intrinsically related to an unfavorable body composition profile, characterized by a prevalence of sarcopenic obesity where an excess of adipose tissue (39.67%) and a critical deficiency of skeletal muscle mass (20.50 kg) coexist.

The study confirms that Body Mass Index (BMI) alone is an insufficient indicator to predict functional health, since the dispersion analysis showed that the increase in body weight is exclusively linked to fat gain ($R^2 = 0.211$) and not to protective musculature ($R^2 = 0.033$), which aggravates metabolic risk. The presence of extreme values of muscle fragility, with alarming minimums of 2.6 kg, reveals a high physical vulnerability that compromises the autonomy and functional capacity of individuals within their social environment. Finally, given the classification of accelerated aging identified through anthropometric indicators, it is concluded that the implementation of comprehensive intervention programs that prioritize

body recomposition, muscle strengthening, and adequate nutrition is imperative to mitigate the negative impact on health and ensure healthy aging in the Ecuadorian rural population.

REFERENCES

- Mesnage, R. (2025). Environmental health is ignored in longevity research. *Antioxidants*, *14*(4), 421. <https://doi.org/10.3390/antiox14040421>
- Guo, J., Huang, X., Dou, L., Yan, M., Shen, T., Tang, W., et al. (2022). Aging and aging-related diseases: From molecular mechanisms to interventions and treatments. *Signal Transduction and Targeted Therapy*, *7*(1), 391. <https://doi.org/10.1038/s41392-022-01251-0>
- Maldonado, E., Morales-Pison, S., Urbina, F., & Solari, A. (2023). Aging hallmarks and the role of oxidative stress. *Antioxidants*, *12*(3), 651. <https://doi.org/10.3390/antiox12030651>
- Proal, A. D., & VanElzakker, M. B. (2025). Pathogens accelerate features of human aging: A review of molecular mechanisms. *Ageing Research Reviews*, *112*, 102865. <https://doi.org/10.1016/j.arr.2025.102865>
- Li, Y., Tian, X., Luo, J., Bao, T., Wang, S., & Wu, X. (2024). Molecular mechanisms of aging and anti-aging strategies. *Cell Communication and Signaling*, *22*(1), 285. <https://doi.org/10.1186/s12964-024-01663-1>
- McHugh, D., Durán, I., & Gil, J. (2025). Senescence as a therapeutic target in cancer and age-related diseases. *Nature Reviews Drug Discovery*, *24*(1), 57–71. <https://doi.org/10.1038/s41573-024-01074-4>
- Bruserud, Ø., Vo, A. K., & Rekvam, H. (2022). Hematopoiesis, inflammation and aging: The biological background and clinical impact of anemia and increased C-reactive protein levels on elderly individuals. *Journal of Clinical Medicine*, *11*(3), 706. <https://doi.org/10.3390/jcm11030706>
- Forrester, S. N., Baek, J., Hou, L., Roger, V., & Kiefe, C. I. (2024). A comparison of five measures of accelerated biological aging and their association with incident cardiovascular disease: The CARDIA study. *Journal of the American Heart Association*, *13*(8), e032847. <https://doi.org/10.1161/JAHA.123.032847>
- Zhang, X., Yan, Y., Liu, Y., Wang, Z., Jiang, Y., Zhang, S., et al. (2025). Association of biological aging acceleration transitions and burdens with incident cardiovascular disease: Longitudinal insights from a national cohort study. *BMC Medicine*, *23*(1), 347. <https://doi.org/10.1186/s12916-025-04177-w>
- Jiang, M., Tian, S., Liu, S., Wang, Y., Guo, X., Huang, T., et al. (2024). Accelerated biological aging elevates the risk of cardiometabolic multimorbidity and mortality. *Nature Cardiovascular Research*, *3*(3), 332–342. <https://doi.org/10.1038/s44161-024-00438-8>
- Zeng, Z., Yu, C., Chen, R., Li, Z., Wang, P., Wang, X., et al. (2025). Biological aging and incident cardiovascular diseases in individuals with diabetes: Insights from a large prospective cohort study. *Cardiovascular Diabetology*, *24*(1). <https://doi.org/10.1186/s12933-025-02855-w>
- Gao, X., Geng, T., Jiang, M., Huang, N., Zheng, Y., Belsky, D. W., et al. (2023). Accelerated biological aging and risk of depression and anxiety. *Nature Communications*, *14*(1), 2277. <https://doi.org/10.1038/s41467-023-38013-7>

- Robinson, O., Chadeau-Hyam, M., Karaman, I., Climaco Pinto, R., Ala-Korpela, M., Handakas, E., et al. (2020). Determinants of accelerated metabolomic and epigenetic aging in a UK cohort. *Aging Cell*, *19*(6), e13149. <https://doi.org/10.1111/accel.13149>
- Qin, T., Chen, T., Ma, R., Li, H., Li, C., Zhao, J., et al. (2024). Stress hormones: Unveiling the role in accelerated cellular senescence. *Aging and Disease*, *16*(4), 1946–1970. <https://doi.org/10.14336/AD.2024.0262>
- Pandics, T., Major, D., Fazekas-Pongor, V., Szarvas, Z., Peterfi, A., Mukli, P., et al. (2023). Exposome and unhealthy aging: Environmental drivers from air pollution to occupational exposures. *GeroScience*, *45*(6), 3381–3408. <https://doi.org/10.1007/s11357-023-00913-3>
- Di Ciaula, A., & Portincasa, P. (2020). The environment as a determinant of successful aging or frailty. *Mechanisms of Ageing and Development*, *188*, 111244. <https://doi.org/10.1016/j.mad.2020.111244>
- Dalecka, A., Bartoskova Polcrova, A., Pikhart, H., Bobak, M., & Ksinan, A. J. (2024). Living in poverty and accelerated biological aging. *BMC Public Health*, *24*(1), 458. <https://doi.org/10.1186/s12889-024-17960-w>
- Ibáñez de Opakua, A., Conde, R., de Diego, A., Bizkarguenaga, M., Embade, N., Lu, S. C., et al. (2025). Metabolomic-based aging clocks. *NPJ Metabolic Health and Disease*, *3*(1), 35. <https://doi.org/10.1038/s44324-025-00078-x>
- Ou, M. Y., Zhang, H., Tan, P. C., Zhou, S. B., & Li, Q. F. (2022). Adipose tissue aging: Mechanisms and therapeutic implications. *Cell Death & Disease*, *13*(4), 300. <https://doi.org/10.1038/s41419-022-04752-6>
- Bellelli, G., Triolo, F., Ferrara, M. C., Deiner, S. G., Morandi, A., & Cesari, M. (2024). Delirium and frailty in older adults. *Journal of Internal Medicine*, *296*(5), 382–398. <https://doi.org/10.1111/joim.20014>
- Korchazhkina, N. B., Mikhailova, A. A., Kotenko, K. V., Nagornyev, S. N., Frolkov, V. K., & Badimova, A. V. (2024). Systemic mechanisms of premature aging and their correction by complex physiotherapy application. *Voprosy Kurortologii, Fizioterapii i Lechebnoi Fizicheskoi Kultury*, *101*(6), 60–70. <https://doi.org/10.17116/kurort202410106260>
- Ventura, L., Cano, A., Morrone, M., Martinez, G., Boi, A., Catta, M. G., et al. (2025). Developing a quantitative estimate of muscle age acceleration by a novel phenotypic clock. *Aging*, *17*(6), 1466–1483. <https://pubmed.ncbi.nlm.nih.gov/40500122/>
- Toyoshima, K., Seino, S., Tamura, Y., Ishikawa, J., Chiba, Y., & Ishizaki, T. (2022). Difference between physical fitness age and chronological age. *Journal of Nutrition, Health & Aging*, *26*(5), 501–509. <https://doi.org/10.1007/s12603-022-1786-8>
- Wang, C., Zheng, R., Song, W., Sun, X., Du, X., & Lu, C. (2025). Sarcopenic obesity and biological aging. *BMC Public Health*, *25*(1), 2226. <https://doi.org/10.1186/s12889-025-23424-6>
- Gustafsson, T., & Ulfhake, B. (2021). Sarcopenia: What is the origin of this aging-induced disorder? *Frontiers in Genetics*, *12*, 688526. <https://doi.org/10.3389/fgene.2021.688526>

- Luo, Y., Fujiwara-Tani, R., Kawahara, I., Goto, K., Nukaga, S., Nishida, R., et al. (2024). Cancerous conditions accelerate skeletal muscle aging. *International Journal of Molecular Sciences*, 25(13), 7060. <https://doi.org/10.3390/ijms25137060>
- Sasako, T., Umehara, T., Soeda, K., Kaneko, K., Suzuki, M., Kobayashi, N., et al. (2022). Deletion of skeletal muscle Akt1/2 causes osteosarcopenia and reduces lifespan. *Nature Communications*, 13(1), 5655. <https://doi.org/10.1038/s41467-022-33008-2>
- Damluji, A. A., Alfaraidhy, M., AlHajri, N., Rohant, N. N., Kumar, M., & Al Malouf, C. (2023). Sarcopenia and cardiovascular diseases. *Circulation*, 147(20), 1534–1553. <https://doi.org/10.1161/CIRCULATIONAHA.123.064071>
- Knoedler, S., Schliermann, R., Knoedler, L., Wu, M., Hansen, F. J., Matar, D. Y., et al. (2023). Impact of sarcopenia on outcomes in surgical patients. *International Journal of Surgery*, 109(12), 4238–4262. <https://doi.org/10.1097/JS9.0000000000000688>
- Zhang, J., Guo, J., Zhang, J., Liu, H., Zhou, L., Cheng, C., et al. (2025). Biological age as a mediator between gut microbiota and sarcopenia. *Frontiers in Immunology*, 16, 1552525. <https://doi.org/10.3389/fimmu.2025.1552525>
- Swan, L., Warters, A., & O’Sullivan, M. (2022). Socioeconomic disadvantage and sarcopenia. *Journal of Frailty & Aging*, 11(4), 398–406. <https://doi.org/10.14283/jfa.2022.32>
- Selvais, C. M., Davis-López de Carrizosa, M. A., Versele, R., Dubuisson, N., Noel, L., Brichard, S. M., et al. (2024). Challenging sarcopenia: Exploring AdipoRon. *Antioxidants*, 13(9), 1073. <https://doi.org/10.3390/antiox13091073>
- Wang, T., Zhou, D., & Hong, Z. (2025). Sarcopenia and cachexia: Molecular mechanisms and therapeutic interventions. *MedComm*, 6(1). <https://doi.org/10.1002/mco2.70030>
- He, P., Du, G., Qin, X., Li, Z., & Li, Z. (2023). Reduced energy metabolism contributing to aging of skeletal muscle. *Life Sciences*, 323, 121619. <https://doi.org/10.1016/j.lfs.2023.121619>
- Antoun, E., Garratt, E. S., Taddei, A., Burton, M. A., Barton, S. J., Titcombe, P., et al. (2022). Epigenome-wide association study of sarcopenia. *Journal of Cachexia, Sarcopenia and Muscle*, 13(1), 240–253. <https://doi.org/10.1002/jcsm.12876>
- Cai, Y., Han, Z., Cheng, H., Li, H., Wang, K., Chen, J., et al. (2024). Impact of ageing mechanisms on musculoskeletal diseases. *Frontiers in Immunology*, 15, 1405621. <https://doi.org/10.3389/fimmu.2024.1405621>
- Afraz, E. S., Hoseinikhah, S. A., & Moradikor, N. (2025). Recent advances in aging-related diseases. *Aging and Disease*, 16(4), 1785–1792. <https://doi.org/10.14336/AD.2025.10618>
- Laviano, A. (2023). Sarcopenia, biological age and treatment eligibility in cancer. *Current Opinion in Clinical Nutrition and Metabolic Care*, 26(1), 59–63. <https://doi.org/10.1097/MCO.0000000000000888>
- Kamarulzaman, N. T., & Makpol, S. (2025). The link between mitochondria and sarcopenia. *Journal of Physiology and Biochemistry*, 81(1), 1–20. <https://doi.org/10.1007/s13105-024-01062-7>